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Interpreting Urban Carbon Monoxide Concentrations by a Computerized COHb Model

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16. ABSTRACT

A practical, inexpensive computer model for estimating the level of blood carboxyhemoglobin (percent COHb) as a function of time for measured carbon monoxide concentrations (ppm CO) was developed from published studies on the assimilation of CO into the blood of human subjects. The model was designed to consider realistically the dynamic characteristics of urban CO concentrations measured continuously at air monitoring stations, and it was applied to a year's CO data measured at the San Jose CA air monitoring station (8760 hourly values).

According to the model, the National Ambient Air Quality Standards (NAAQS) for CO sometimes were violated in San Jose without exceeding 2% COHb, as well as the converse: 2% COHb was exceeded without violating the standards. The model's estimated COHb levels also provided an advance warning of impending violations of the 8-hr CO NAAQS, and analysis of the model's response to CO "spikes" suggests that averaging periods as short as 10 or 15 minutes are necessary to serve completely the dynamic characteristics of ambient CO monitoring data. These findings suggest that the margin of safety included in the current CO NAAQS would not be the same if the actual time variation of measured CO concentrations is taken into account.

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Interpreting Urban Carbon Monoxide Concentrations by Means of a Computerized Blood COHb Model

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A practical, inexpensive computer model for estimating the level of blood carboxyhemoglobin (percent COHb) as a function of time for measured carbon monoxide concentrations (ppm CO) was developed from data from published studies on the assimilation of CO into the blood of human subjects. The model was designed to consider more realistically the *dynamic* characteristics of urban CO concentrations measured continuously at air monitoring stations, and it was applied to a year's CO data measured at the San José CA, air monitoring station (8760 hourly values).

The results indicate that the model can be used by local air pollution control agencies to calculate and print out *estimated* COHb levels alongside continuous CO concentration data. According to the model, the National Ambient Air Quality Standards (NAAQS) for CO sometimes were violated in San José without exceeding 2% COHb, as well as the converse: 2% COHb was exceeded without violating the standards. The model's estimated COHb levels also provided an advance warning of impending violation of the 8-hr CO NAAQS, and analysis of the model's response to CO "spikes" suggests that averaging periods as short as 10 or 15 minutes are necessary to preserve completely the dynamic characteristics of ambient CO monitoring data. These findings suggest that the margin of safety included in the current CO NAAQS, would not be the same if the actual time variation of measured CO concentrations is taken into account.

Carbon monoxide (CO) is a hazard to man because it tends to be assimilated by the blood molecule, hemoglobin, interfering with the transport of oxygen from the lungs to all parts of the body. In the lung capillaries, the hemoglobin molecule combines with oxygen. This hemoglobin-oxygen complex is carried through the blood stream and when it reaches a body cell that requires oxygen the bond is broken and the oxygen is released for use by the cell.

The oxidation process which occurs within the cells ordinarily produces some CO. This CO combines with hemoglobin to form COHb for a return trip to the lungs. There the CO is released and exhaled from the body. As a result, normal human blood always contains an equilibrium concentration of COHb. This internal value varies from individual to individual, but it is on the order of 0.5%.[1]

Peterson and Stewart[2] have exposed human volunteers to a variety of different CO concentrations for periods ranging from ½ to 24 hr. Using a regression approach, they derive the following empirical relationship for blood carboxyhemoglobin as a function of CO concentration and exposure time:

$$\log_{10} y = 0.85735 \log_{10} x + 0.62995 \log_{10} t - 2.29519 \quad (1)$$

where y = percent COHb
x = CO concentration (ppm)
t = time (min)

The National Ambient Air Quality Standards (NAAQS) for CO, which are based on this relationship, are designed to "protect against the occurrence of carboxyhemoglobin levels above two percent."[3] The NAAQS specify the following concentrations and averaging times: 35 ppm CO for one hour; 9 ppm CO for 8 hours. Although designed to protect against the occurrence of 2% COHb in an average person, these standards are intended to "include an adequate margin of safety." Substitution of these values into Eq. (1), for example, gives 1.4% COHb for the 35 ppm standard and 1.6% COHb for the 9 ppm standard—significantly below the 2% level. This implies "safety factors" of 1.43 and 1.25, respectively.

The Peterson and Stewart model of Eq. (1) is basically a *static model*. It gives the COHb level for any time period t

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provided that the CO concentration x remains *constant* over this period. Actually, observed ambient CO concentrations in urban areas usually vary from hour to hour and from minute to minute. Would a *dynamic model*, which properly takes into account these variations of CO with time, predict COHb levels above 2% without exceeding the federal CO standards? That is, do CO concentrations vary over a 24-hr period in such a manner that they can violate the physiological basis for the standards without violating the standards themselves?

To answer this question, we developed a more flexible COHb model that is better suited to interpreting the dynamic CO concentrations recorded continuously by urban air monitoring stations. Then we applied the model to a year of measured CO data and examined the results.

Development of the Model

Although it is possible to develop more complex models of the blood CO assimilation process, there does not appear to be sufficient data available to validate such models. For our purposes, the most reasonable approach is to construct the simplest possible model that can be readily supported by existing research data. Thus, we attempted to synthesize the findings of a number of different research investigators into one general formulation.

Steady State Response

A serious limitation of the model of Peterson and Stewart is that it does not possess an asymptote. There is widespread agreement in the literature[2,4-7] that, after human subjects are exposed to a particular CO concentration for a long period, blood COHb reaches a constant value and remains at this value. This asymptotic property is seen clearly in experimental results obtained by Smith[4] (Figure 1), who placed seven non-smoking males in a large room filled with a uniform concentration of 30 ppm CO. The CO concentration was maintained by mixing a small quantity of pure CO with the room's air and circulating the air with a fan. The men stayed in the room for 24 hr, engaging in light physical activity such as reading, talking, and card playing.

Coburn[5] has developed theoretical relationships for the major physiological variables that determine blood COHb levels both under steady state and transient conditions. He includes the following parameters in his model: internal rate of CO production, alveolar ventilation, diffusing capacity of the lung, mean oxygen tension in the pulmonary capillaries, and CO concentration in the inhaled air. His approach treats total COHb as the sum of two independent components: (1) *exogenous* COHb resulting from inhalation of air containing CO, and (2) *endogenous* COHb generated internally by the body. That is,

$$\mathrm{COHb_{total}} = \mathrm{COHb_{exogenous}} + \mathrm{COHb_{endogenous}} \qquad (2)$$

His model shows good agreement with his experiments, in which blood COHb samples are taken from selected individuals while the air is monitored for CO concentration.

In Coburn's analysis, COHb concentration at equilibrium $\bar{y}_i$ is related to x_i in a linear fashion, where x_i is constant with time. This linear relationship is consistent with findings reported elsewhere in the literature.[6,7] Although Peterson[8] fits a quadratic relationship to his data, a linear model appears to fit the data almost as well for CO concentrations under 100 ppm. If β denotes the endogenous COHb component, the equilibrium value of blood COHb—using a linear model—would be related to the CO concentration as follows:

$$\left.\begin{aligned} y_i &= \alpha x_i + \beta \\ 0 \le x_i &\le 100 \end{aligned}\right\} \qquad (3)$$

Goldsmith[6] states that the California State Department of Public Health has derived, from published data, a value equivalent to $\alpha = 0.16$ for CO concentrations under 100 ppm. McIlvaine[7] cites a value equivalent to $\alpha = 0.181$, which he derives from empirical studies. Analysis of Smith's experimental data leads to a value of $\alpha = 0.15$. Coburn[5] arrives at the same value. These results suggest that α lies between 0.15 and 0.18. We choose $\alpha = 0.15$ for our model.

A reasonable value for β, the endogenous level of blood COHb, is $\beta = 0.5\%$, the value cited in the federal air quality criteria for CO.[1] Treatment of endogenous blood COHb as a constant is an oversimplification, however, because it may vary from individual to individual and depends on many factors, including level of physical activity and lung capacity. Nevertheless, there does not appear to be enough data to justify a more complex treatment, and a single "mean" endogenous COHb value is satisfactory for the purposes for which our model is intended.

Transient Response

Smith's data (Figure 1) indicate the nature of the increase of blood COHb with time for nonsmokers exposed to 30 ppm CO and engaged in light physical activity. He describes the characteristic shape of the curve of COHb concentration with respect to time as follows:

> . . .during continuous exposure to 30 ppm carbon monoxide, about 60% of the equilibrium concentration of carboxyhemoglobin is attained within a matter of two hours, while 80% is attained within a four hour period. The remaining 20% is achieved relatively slowly during the next eight or more hours.[4]

If a smooth curve which conforms to Smith's description is plotted, it is found to be a true exponential, with a time constant of 2.49 hr:

$$y(t) = 5 - 4.5\, e^{-t/2.49} \qquad (4)$$

where $y(t)$ is blood COHb (%) as a function of time (hr) and e is the base of the natural logarithm. We have plotted this curve in Figure 1. Although this function differs from that selected by Peterson and Stewart, curves of the two functions are similar in shape for a constant CO concentration.

The curve represented by Eq. (4) has the property that the rate of change of $y(t)$ is proportional to the difference between $\bar{y}_i$, the final value, and $y(t)$, and this can be represented as a first order, linear differential equation:

$$\tau \frac{dy(t)}{dt} = \bar{y}_i - y(t) \qquad (5)$$

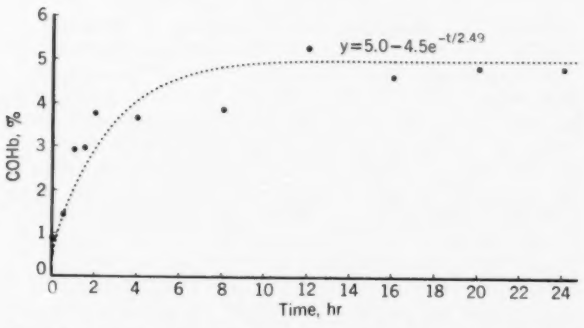
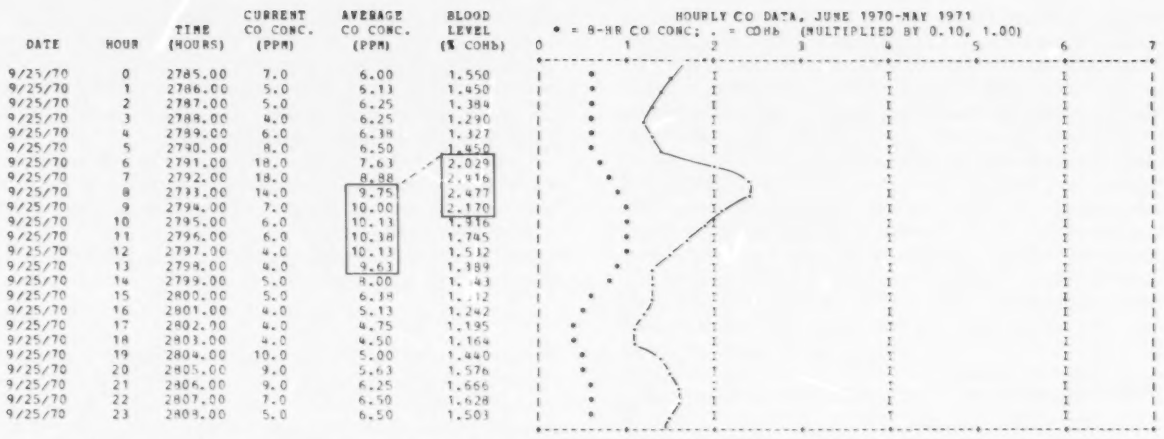


Figure 1. Concentrations of COHb as a function of time in 7 male nonsmokers as reported by Smith[4] along with exponential model.



DATE	HOUR	TIME (HOURS)	CURRENT CO CONC. (PPM)	AVERAGE CO CONC. (PPM)	BLOOD LEVEL (% COHb)
9/25/70	0	2785.00	7.0	6.00	1.550
9/25/70	1	2786.00	5.0	6.13	1.450
9/25/70	2	2787.00	5.0	6.25	1.384
9/25/70	3	2788.00	4.0	6.25	1.290
9/25/70	4	2789.00	6.0	6.38	1.327
9/25/70	5	2790.00	8.0	6.50	1.450
9/25/70	6	2791.00	18.0	7.63	2.029
9/25/70	7	2792.00	18.0	8.88	2.916
9/25/70	8	2793.00	14.0	9.75	2.477
9/25/70	9	2794.00	7.0	10.00	2.170
9/25/70	10	2795.00	6.0	10.13	1.916
9/25/70	11	2796.00	6.0	10.38	1.745
9/25/70	12	2797.00	4.0	10.13	1.532
9/25/70	13	2798.00	4.0	9.63	1.389
9/25/70	14	2799.00	5.0	8.00	1.243
9/25/70	15	2800.00	5.0	6.38	1.212
9/25/70	16	2801.00	4.0	5.13	1.242
9/25/70	17	2802.00	4.0	4.75	1.195
9/25/70	18	2803.00	4.0	4.50	1.164
9/25/70	19	2804.00	10.0	5.00	1.440
9/25/70	20	2805.00	9.0	5.63	1.576
9/25/70	21	2806.00	9.0	6.25	1.666
9/25/70	22	2807.00	7.0	6.50	1.628
9/25/70	23	2808.00	5.0	6.50	1.503

Figure 2. Computer printout for September 25, 1970, showing the 2% COHb level exceeded in the morning rush hour (6–9 A.M.) two hours before the federal air quality standard of 9 ppm for 8 hr is violated.

where τ is the time constant, and d/dt denotes the first derivative with respect to time. Coburn's model also embodies a linear differential equation of the same form as Eq. (5). Eq. (4) represents one solution of the differential equation given by Eq. (5). It is known as the "step response" of the model, because x_i may be viewed as a "step" which is zero prior to $t = 0$, but which suddenly has a value applied at time $t = 0$, remaining constant thereafter.[9]

General Model

Combining Eq. (3) and Eq. (5), a general model is developed relating blood COHb to the inspired CO concentration:

$$\left.\begin{aligned} \tau \frac{dy}{dt} + y - \beta &= \alpha x \\ 0 \le x &\le 100 \end{aligned}\right\} \qquad (6)$$

where x = CO concentration
y = blood COHb level

For simplicity, $x(t)$ and $y(t)$ are denoted here simply as x and y, respectively. Eq. (6) embodies both the transient and steady state response in one formulation. Based on Smith's data, we choose $\tau = 2.49$ hr, which corresponds to a "half-time" of 1.73 hr. An implicit assumption of this model is that the coefficients are constant and that the system being modeled is linear. This implies that the time constant for CO absorption into the blood is the same as for CO exhalation. In actuality, this may be slightly in error, but there is little data available to support a more complex assumption. In developing this model, we also found it necessary to assume that the level of physical activity remains constant and that the individuals are engaged in light physical activity.

When subjects are at rest (lying down), the rate of CO uptake decreases, and the time constant increases to 3–4 hr, which corresponds to a half-time of 2–3 hr. In a recent *JAPCA* Critical Review article, Ferris[10] analyzed the CO standard using a CO uptake rate for a person at rest on the order of $\tau = 3.5$ hr. He concluded that the present 1-hr and 8-hr standards seem quite adequate for persons at rest and should be protective even for exercising individuals. The assumption that the standards protect against the occurrence of 2% COHb for active individuals is tested in the following sections by applying our dynamic model to actual urban CO concentration data.

Application of the Model

To apply the model to urban air quality data, we obtained a year's CO data (8760 hourly values) from a modern, well-equipped air monitoring station operated in San José, California, by the Bay Area Air Pollution Control District.

In applying this model, each successive hourly CO value can be treated as an independent step function, provided that the COHb level at the end of the preceding hour is taken into account. If we denote the exogenous COHb level for a particular hour as $\hat{y}_1$, then $\hat{y}_2$, the exogenous COHb level for the next hour, is a simple linear function of $\hat{y}_1$ and x_2. This is because, for hourly averages, the duration of exposure has a value of 1.0 hr for each CO entry. For $\tau = 2.49$ hr:

$$\begin{aligned} \hat{y}_2 &= (\alpha x_2 - \hat{y}_1)(1 - e^{-1.0/2.49}) + \hat{y}_1 \\ &= 0.331\, \alpha x_2 + 0.669\, \hat{y}_1 \end{aligned} \qquad (7)$$

The computer algorithm developed for this relationship can be written as a general difference equation for any time period n, where $\alpha = 0.15$, $\beta = 0.5$, and total COHb y_n is given as the sum of both the exogenous and endogenous COHb components:

$$\left.\begin{aligned} \hat{y}_n &= 0.0496\, x_n + 0.669\, \hat{y}_{n-1} \\ y_n &= \hat{y}_n + 0.5 \\ (n &= 1, 2, 3, \ldots) \end{aligned}\right\} \qquad (8)$$

In Eq. (8), x_n represents each of the 8760 values occurring over the year. Because this equation is a "difference" equation, it gives current COHb as a function of the present CO concentration and the previous COHb level (beginning at $n = 1$). Its current estimate of blood COHb depends, therefore, not only on the current CO concentration but also on the history of CO concentrations. Initial conditions were set as $\hat{y}_o = 0$ for $n = 1$, and the computer programs were written to retain the value of the preceding hour whenever a missing CO value was encountered (only 2% of the 8760 values were missing).

Because of its inherent simplicity, Eq. (8) can readily be programmed on a computer, minicomputer, or hand calculator and can analyze continuous CO concentrations quite inexpensively.

Results

For the year-long data set, the results for San José were printed graphically on the line printer, giving the estimated

COHb level for each hour and a total of 80 pages of computer printout. Figure 2 shows an example of the printout for one 24 hr period—September 25, 1970. In this figure, the first column gives the date on which the CO concentration at the air monitoring station was recorded; the second column shows the hour of the day (starting at 12:00 midnight); the third column shows the time in hours elapsed since the beginning of the data run. The fourth column ("Current CO") shows the hourly average CO concentration (input to the model); the next column gives the 8 hr average CO concentration (averaged over the previous 8 hr); and, finally, the last column gives percent COHb as predicted by the model. This page of the printout begins at the 2785*th* hour of the year; the COHb level at that time results from the 2784 hourly CO values entered since June 1, 1970, the beginning of the data set.

According to the model, the COHb level on September 25 exceeded 2% during the morning traffic rush period (between 6:00 A.M. and 10:00 A.M.), while the 8 hr average CO concentration exceeded the 9 ppm federal CO standard from 8:00 A.M. to noon. Thus, the model predicted that the 2% COHb level was exceeded 2 hr before the federal CO standard was actually violated.

This figure also illustrates how the model can be used by air pollution control agencies to print out estimated COHb levels alongside measured CO concentrations. Such a printout should provide a more accurate indication of the potential health-related impact of CO concentrations measured in the immediate vicinity of air monitoring stations than the raw CO data. In addition, we found that high estimated COHb levels generally occurred several hours before the 8 hr CO standard was violated, enabling the model to give advance predictions of violations of the NAAQS.

To facilitate our comparison of federal CO standards in relation to the 2% COHb level, we have found it convenient to introduce two terms: (1) *violation*, which refers to a violation of the CO NAAQS, and (2) *exceedance*, which refers to an instance in which the model says COHb exceeds 2%. Over the entire year of data in San José (June 1, 1970, to May 31, 1971), 25 separate COHb exceedances occurred, according to the model. By comparison, there were 29 violations of the 9 ppm federal CO standard, and no violations of the 35 ppm CO standard. Of the 25 instances in which COHb levels exceeded 2%, there were *two* cases in which the federal CO standard of 9 ppm *was not* violated. Of the 29 violations of federal CO standards, there also were 6 instances of the converse: violation of federal CO standards without a 2% COHb exceedance.

These results have implications for the *margin of safety* which is supposed to be built into the federal NAAQS. Assuming safety factors as large as the 1.43 and 1.25 discussed earlier, we would expect to find *no instances* in which the 2% COHb level is exceeded without the standard being exceeded also. Yet, according to the model, such instances occurred for this data set in 2 out of 25, or 8%, of the COHb exceedances.

Response of Model to CO Spikes

Although air monitoring instruments such as the infrared CO analyzer provide a continuous record of the CO concentration on an ink trace, it is customary for air pollution control agencies routinely to convert these traces into hourly average values and to store the data as such. These traces sometimes contain sharp peaks of short duration, or "spikes," particularly if the monitoring station is located near heavy traffic or on congested streets. Sometimes, these spikes are as high as 20 to 30 ppm, with occasional values of 60 ppm. If only the average value is known, however, there is no way to determine that such spikes have or have not occurred. How does the presence of such spikes affect blood COHb? This problem can be stated another way: suppose, for a typical measurement, we know that the average value of the CO concentration is 10 ppm, but that is all we know about it. How will the shape of the function—its "peakedness" (amplitude in relation to duration)—affect predicted blood COHb?

To consider this problem, we examine the response of the model to a rectangular input of the form shown in Figure 3. For simplicity, only the exogenous component of blood COHb is considered. The maximum exogenous blood COHb resulting from the rectangular input is given as follows, where Δt is the duration of the rectangular input, and x is its amplitude:

$$\hat{y} = \alpha\, x\, (1 - e^{-\Delta t/\tau}) \tag{9}$$

We can increase the peakedness of a function by making the duration successively smaller as the amplitude becomes successively larger; if, however, the area under the function remains the same, the average value over the 1 hr period also will, by definition, remain the same. In going from a CO concentration of 10 ppm for 1 hr to 50 ppm for $\frac{1}{5}$ hr (12 min), as in Figures 3(a), 3(b), and 3(c), the predicted exogenous blood COHb changes from 0.496% to 0.580%.

Suppose this process is continued indefinitely. To keep the average value constant, the product $x\,\Delta t$ must be held constant. Substituting $\Delta t = \text{const}/x$ into Eq. (9), where "const" denotes the area under the input, one obtains a general relationship for the maximum exogenous COHb level resulting from a rectangular input function of any amplitude x:

$$\hat{y} = \alpha\, x\, (1 - e^{-\text{const}/x\tau}) \tag{10}$$

As the amplitude becomes increasingly large, x approaches infinity, and, expanding Eq. (10) by means of a Taylor series, all terms involving x in the denominator approach zero:

$$\lim_{x \to \infty} \left\{ \alpha\, x \left(1 - \left[1 - \frac{\text{const}}{x\,\tau} + \frac{1}{2!}\left(\frac{\text{const}}{x\,\tau}\right)^2 - \frac{1}{3!}\left(\frac{\text{const}}{x\,\tau}\right)^3 + \dots \right] \right) \right\} = \frac{\alpha}{\tau}\,(\text{const}) \tag{11}$$

For an input with an average value of 10 ppm, and the constants embodied in our model, Eq. (11) gives 0.602% COHb. This input, an infinitely tall peak of zero duration and finite area, commonly is called a *delta function*. In moving from one extreme to the other—the case of minimum peakedness (a uniform value over the hour) to the case of maximum peakedness (an infinitely tall peak), the maximum predicted (exogenous) blood COHb changed from 0.496% to 0.602%, an increase of 21%. Without additional information on the shape of the CO input function during the hour, one cannot determine the exact level of COHb which will result, but it must lie within these two extremes. It also can be shown that 21% is the maximum error which can occur, regardless of the average value of the rectangular input. This finding pertains only to the exogenous component of blood COHb; when the endogenous component is added, the overall error would, of course, be less than 21%.

All things considered, this error does not appear too great. However, if tall peaks are present in the CO data of a given air monitoring station, as is often the case, these results suggest that CO concentrations should be reported on averaging periods of less than 1 hr. Otherwise, COHb levels derived from these CO concentrations will be *underestimated*—with a maximum possible error of 21%. Averaging periods of 10 or 15 min would appear suitable.

It is essential, of course, that the true average value be obtained during the period and not just the average of a number of instantaneous values read from the chart at fixed intervals, as is done by the automated equipment in operation at some air monitoring stations.

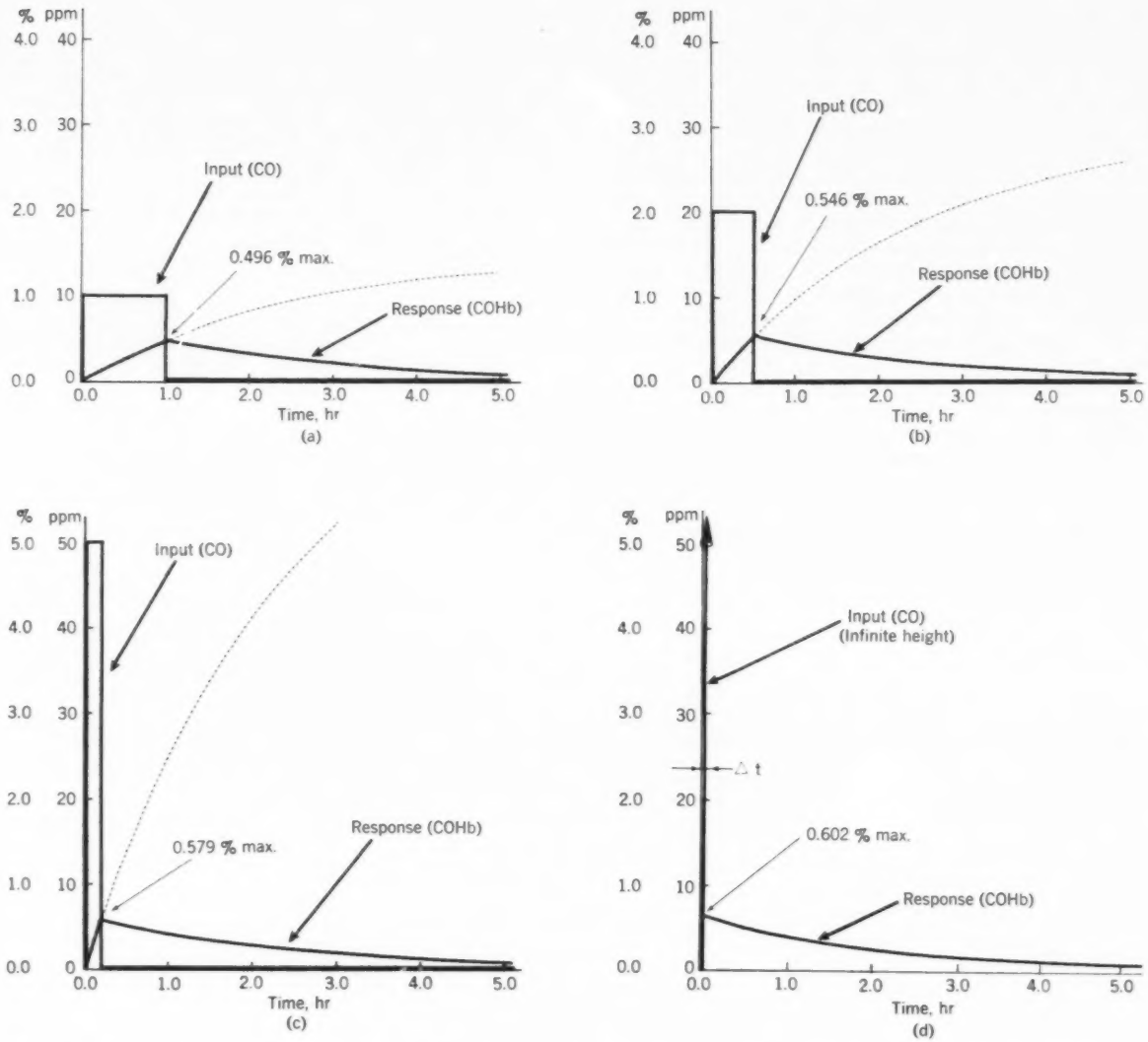


Figure 3. COHb model response to rectangular CO inputs all having the same area of 10 ppm-hr, but each having different magnitude: (a) 10 ppm, (b) 20 ppm, (c) 50 ppm, (d) infinity (delta function).

At some monitoring installations, it is common practice to insert a large bottle in the intake lines to "dampen out" sharp peaks before they reach the instrument. While this practice may seem reasonable at first glance, it has the unfortunate effect of adding a poorly defined, and often nonlinear, filter to the entire CO measurement system. Because of the uncontrolled distortion it may introduce, this practice—in the authors' view—is very undesirable and should be avoided. Filtering, if any, should be done by mathematical manipulations of the computer once the measurements are made; such operations can be carefully designed and maintained under full control of the data analyst.

The model described in this paper offers one example of a set of mathematical manipulations the data analyst may wish to perform. It "filters out" spikes in a clearly defined manner, producing a result that has potential physical meaning—estimated blood COHb.

Summary

This work has concentrated on development of a practical, inexpensive COHb model for use in interpreting carbon monoxide concentrations measured routinely at urban air monitoring stations. The basic equations, along with their constants, were derived from experiments with human subjects which appear in the literature. The model was applied using existing monitoring data in an actual city.

The results imply that the "margin of safety" embodied in federal air quality standards may be smaller than commonly believed. According to the model, it is possible for actual CO concentrations to bring out blood COHb levels in excess of 2% COHb—the value on which the federal standards are based—*without* violating the standards themselves. In a year of data (8760 hourly average values), there were 25 separate instances in which 2% COHb levels were exceeded; in two of

these cases, or 8%, the federal CO standards were not violated. Thus, when the actual temporal variation of CO concentrations is taken into account, the model suggests that current NAAQS do not always protect against the occurrence of 2% COHb, as some contend. However, this model is very simple and tentative and further study of this problem is recommended.

Because of the simplicity of this model, the necessary computations can easily be carried out by computer, minicomputer, or hand calculator, and state and local air monitoring programs can use the model to print estimated COHb concentrations alongside measured CO concentrations on a routine basis. Used in such a fashion, the model has shown itself able to give advance warning of impending violation of the 8-hr CO NAAQS several hours before such violations occur.

This analysis also suggests that the use of averaging periods as long as 1 hr introduces error into recorded urban CO concentrations. If many sharp CO peaks are present, blood COHb levels derived from these hourly averages can be underestimated by as much as 21%. In general, averaging periods shorter than one hour—i.e., 10 or 15 min—would be desirable for recording atmospheric CO concentrations and are recommended, on the basis of these findings, particularly if intake probes are sufficiently near sources (for example, traffic) to exhibit such fluctuations.

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